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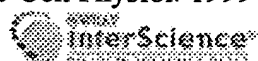
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Clusterin (Apo J) regulates vascular smooth muscle cell differentiation in vitro.

Moulson CL, Millis AJ.

Center for the Study of Comparative Functional Genomics, Department of Biological Sciences, State University of New York at Albany, 12222, USA.

Previously we reported a significant and substantial increase in the synthesis and secretion of clusterin in cultured porcine vascular smooth muscle cells (VSMC) during the time when the VSMC culture modulates from a proliferating monolayer morphology to a nodular cell culture morphology. That in vitro process appears to recapitulate some aspects of in vivo vascular remodeling in response to injury and is facilitated by the presence of a well-developed extracellular matrix. To directly test the hypothesis that clusterin regulates VSMC phenotypic modulation, cultured VSMC were stably transfected with an expression plasmid containing the full-length murine clusterin sequence in antisense orientation. Twenty-four clones were selected on the basis of neomycin resistance and characterized for clusterin expression and culture morphology. In contrast to clone SM-CLU18AS, which expresses a high level of clusterin and forms multicellular nodules, clone SM-CLU13AS expresses a low level of clusterin and does not form nodules even in the presence of a preformed collagen gel. Importantly, clusterin-negative SM-CLU13AS retains the ability to form nodules in an environment containing exogenous clusterin. SM-CLU13AS forms nodules when cultured in Matrigel (which contains clusterin) and in the presence of clusterin-containing conditioned media prepared from nodular SMC cultures or SM-CLU18AS cultures. These results demonstrate that clusterin is required for VSMC nodule formation and suggest that it may play a role in smooth muscle cell reorganization in the vascular wall.

PMID: 10430175 [PubMed - indexed for MEDLINE]

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